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CAN LESIONS IN THE RIGHT BASAL GANGLIA CAUSE APHASIA? CROSSED APHASIA IN A RIGHT-HANDED PATIENT

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ABSTRACT

Background: Aphasia is a common neurocognitive disorder caused by impaired speech and language, with stroke being the most frequent cause. The neuroanatomical mechanism underlying this condition is not yet fully understood.

Case description: This case describes a 74-year-old Caucasian woman admitted with a clinical picture of right total anterior circulation infarct (TACI) and aphasia, scoring 17 on the National Institutes of Health Stroke Scale. Neuroimaging showed a large cortico-subcortical frontotemporoparietal and insular infarct involving the basal ganglia of the right hemisphere and bilateral focal atherosclerotic stenosis on the M1 segment of the middle cerebral artery. There was no left hemispheric lesion or abnormal electric activity on the electroencephalogram. A formal evaluation was compatible with transcortical motor aphasia. The aetiological study revealed atrial fibrillation, and the case was admitted as an ischaemic stroke of undetermined aetiology with two possible causes – intracranial atherosclerotic stenosis or atrial fibrillation.

Conclusion: Our patient fulfilled all the formal criteria for crossed aphasia in dextral (CAD): aphasia, a lesion in the right hemisphere coupled with the structural integrity of the left hemisphere, an established preference for right-hand use without a familial history of left-handedness individuals, and an absence of brain damage in childhood. Our patient's case adds to the evidence that deep structures – alone or in combination with cortical structures – are primarily affected in CAD.

KEYWORDS

Crossed aphasia, crossed aphasia in dextral, aphasia

LEARNING POINTS

- The diagnostic criteria for crossed aphasia in dextral (CAD) are derived from clinical case studies and include aphasia, a lesion in the right hemisphere, a strong preference for using the right hand, the structural integrity of the left hemisphere and no history of brain damage during childhood.
- The right lentiform nucleus was found to be the most frequent anatomical substrate involved in CAD patients, consistent with our case description.
- Our patient experienced transcortical motor aphasia due to a stroke in the right hemisphere, adding to the evidence that in CAD patients, deep structures are primarily affected. In contrast, in left hemispheric lesions, cortical structures seem to be the main culprits.

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INTRODUCTION

Aphasia is a neurocognitive disorder with impaired speech and language. Stroke is the most common cause of aphasia, with an estimated prevalence of 15–50% in all stroke patients[1-4]. Aphasic patients can have a disability in expressive or receptive language. Expressive language defects, also known as non-fluent aphasia, can be further classified as Broca's aphasia, transcortical motor aphasia or global aphasia. Receptive language defects, or fluent aphasia, can be classified into Wernicke's aphasia, transcortical sensory aphasia or conduction aphasia[3]. The type of aphasia can usually be inferred from the lesion pattern in stroke patients due to the anatomy of cerebrovascular territories, and the cortical organisation of speech and language being consistent across individuals[3]. An occlusion in the middle cerebral artery is the main culprit of aphasia in stroke, with affection of the frontotemporal circumvolution and the arcuate fasciculi. Most patients with aphasic syndromes have a lesion in the left hemisphere, which is usually dominant in the human population: 95–99% of right-handed individuals (dextral) have left dominance, and 70% of left-handed individuals (sinistral) also have left hemisphere dominance[5]. Notwithstanding this, an aphasic syndrome can arise from lesions to the right hemisphere. Crossed aphasia is a concept introduced by Bramwell in 1899 and defined as an aphasic syndrome caused by brain damage ipsilateral to the dominant hand[6,7]. This condition occurs in around 30% of stroke patients who are sinistral, but it is extremely rare in dextral patients. Diagnostic criteria for reliable crossed aphasia in dextral (CAD) are aphasia, lesion in the right hemisphere, strong preference for right-hand use without familial history of left-handedness, the structural integrity of the left hemisphere and an absence of brain damage in childhood[8,9].

The neuroanatomical mechanism underlying crossed aphasia in dextral patients is not understood. Proposed explanations include a silent anatomical dysfunction in the left hemisphere, ipsilateral control of the dominant hand, bilateral representation of language and speech functions or a defect in lateralising those functions through the development stage[3,10]. Despite that, the right lentiform nucleus was found to be the most frequent anatomical substrate involved in CAD patients, consistent across several case reports and literature reviews[10].

CASE DESCRIPTION

A 74-year-old Portuguese Caucasian woman with a medical history of hypertension, dyslipidaemia, peripheral artery disease and an episode of monomorphic ventricular tachycardia was admitted to our stroke unit with a clinical picture of total anterior circulation infarct (TACI) manifested by left hemiplegia, left homonymous hemianopsia with right gaze palsy, central facial left palsy, anosognosia and aphasia. Computed tomography (CT) showed a large cortico-subcortical frontotemporoparietal and insular infarct involving the basal ganglia of the right hemisphere *(Fig. 1)*. The CT angiography was relevant for bilateral focal atherosclerotic stenosis on the M1 segment of the middle cerebral artery. Despite scoring 17 on the National Institutes of Health Stroke Scale, no acute-phase treatment was available due to her being last seen 48 hours before admittance with an established lesion having an Alberta stroke program early CT score of 3.

The patient's initial evolution was noteworthy, with a spontaneous haemorrhagic transformation of the ischaemic lesion (classified as haemorrhagic infarction type 2), pneumonia, toxic hepatitis and iatrogenic bradycardia. The aetiological study revealed an atrial fibrillation in the 24-hour Holter evaluation. Therefore, our team admitted an ischaemic stroke of undetermined aetiology with two possible causes – intracranial atherosclerotic stenosis and atrial fibrillation.

After clinical stabilisation, the patient interacted using spontaneous hand gestures and facial expressions. She could execute simple commands while demonstrating extremely poor spontaneous speech with no fluency, and oral apraxia was evident. Comprehension and repetition were preserved, which was also seen in handwriting: she could write repetitive legible letters using the right hand but with no

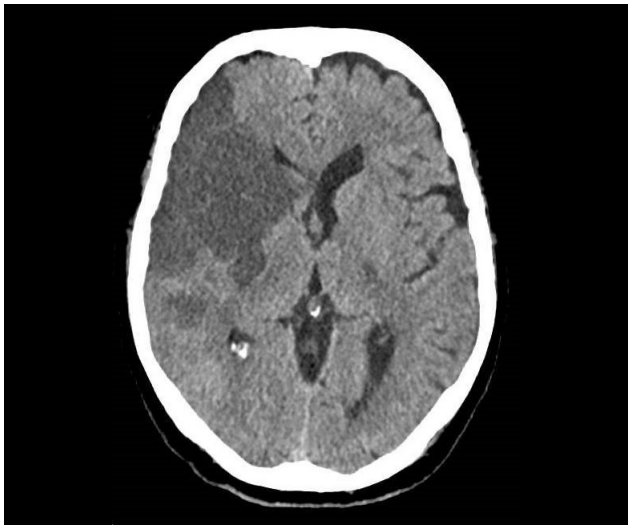

Figure 1. Computed tomography at admission.

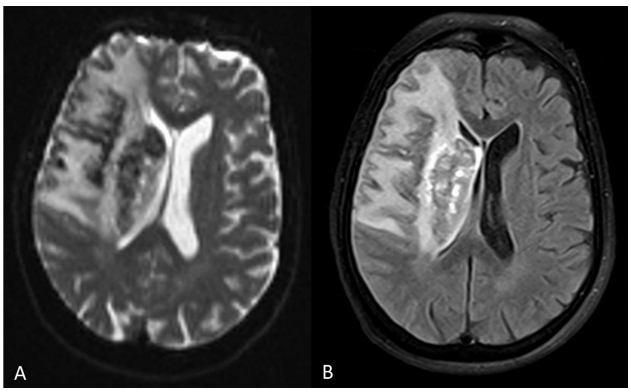

Figure 2. Diffusion magnetic resonance imaging two weeks after admission showing extensive ischaemic stroke and the haemorrhagic component in the basal ganglia of the right hemisphere. A) Diffusion-weighted imaging, B) T2 weighted fluid attenuated inversion recovery.

logical meaning in the words written. A formal evaluation was undertaken by the speech and language therapy team using the Lisbon Aphasia Assessment Battery test, and it was compatible with transcortical motor aphasia. The speech was non-fluent and characterised by perseveration, with difficulty initiating the speech. Simple questions of yes/no were answered correctly sometimes. Naming and comprehension were impaired, while repetition was intact. She was able to understand written words, and writing was impaired. The patient started individual intervention with the speech therapist daily, showing sparse evolution regarding language skills. At the date of discharge, the speech consisted mainly of isolated words and maintained persistence; comprehension skills were better, and the patient could answer yes/no questions correctly, with more initiative for communication and a better functional outcome. Despite bilateral stenosis of the M1 segment, no left hemispheric lesion was found on diffusion magnetic resonance diffusion imaging *(Fig. 2)*, and no abnormal electric activity was found on the electroencephalogram.

The patient was discharged from the stroke unit to the rehabilitation programme one month after admittance. She was formally reassessed three months after the cerebrovascular event, maintaining significant motor impairment with left hemiplegia, minor facial palsy and complete left hemianopsia. Regarding speech and language, she showed little to no evolution. With the help of relatives, we confirmed no familial history of left-handedness, no enforcement of right-hand use in childhood, and the absence of any history suggesting brain damage in infancy.

DISCUSSION

Our patient fulfilled all the formal criteria for CAD. An aphasic syndrome was identified, which was further classified as transcortical motor aphasia. A lesion in the right hemisphere was coupled with the structural integrity of the left hemisphere; there was an established preference for right-hand use without familial history of sinistral individuals and an absence of brain damage in childhood. Our patient also seemed to add to the evidence suggesting a pivotal role for the right lentiform nucleus in this type of aphasia.

Kim et al. described seven patients with CAD secondary to ischaemic stroke[10]. They mapped the neuroanatomical structures involved in these patients: six patients had their lentiform nucleus affected and out of these, three had only their subcortical regions impaired. Our patient's case adds to the evidence that deep structures – alone or in combination with cortical structures – are primarily affected in CAD patients. In contrast, cortical structures are the main culprits in aphasia caused by left hemispheric lesions.

According to the most significant collection of clinical information about CAD, comprising 167 patients, the majority were men (65%) with a mean age of 57[11]. Noteworthy, 61.8% had equivalent written and oral language impairment, and verbal apraxia was present in 45.1% of CAD patients. The latter was consistent with our case.

CONCLUSION

Given the uncommon occurrence of CAD and its stringent criteria, we believe that our patient's case provides valuable insights into this particular condition. Further correlation between clinical information and neuroimaging findings is required to better understand the neuroanatomical pathways affected in CAD.

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